Article

Heart disease in cattle with clinical signs of heart failure: 59 cases

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Abstract — This retrospective study identified clinical signs, underlying cardiac conditions, blood findings, echocardiographic findings, and prognosis for 59 cattle with clinical signs of congestive heart failure. Signalment; history; clinical signs; clinicopathologic, echocardiographic, and radiographic findings; and treatment were determined by reviewing medical records. Follow-up information was obtained by telephone conversation with owners. Most patients were tachycardic (n = 50), and tachypneic (n = 55). Pericarditis of traumatic origin (n = 21), by extension from pleuritis (n = 3), or of idiopathic origin (n = 1) was diagnosed in 25 cases. Other diagnoses were congenital heart defect (n = 13), cardiomyopathy (n = 9), bacterial endocarditis (n = 7), and neoplasm (n = 5). Twelve cases (20%) were discharged. Long-term survival was good in 2 out of 3 cases treated by pericardiostomy. The prognosis is poor in cases of heart failure in cattle and deaths within 1 mo (n = 3) or between 1 to 3 mo after discharge (n = 3) were common in cases for which follow-up was available (n = 8).

Résumé — Les maladies cardiaques chez les bovins avec des signes cliniques d'insuffisance cardiaque : 59 cas. L'objectif de cette étude rétrospective était d'identifier les signes cliniques, les maladies cardiaques sous-jacentes, les résultats des examens sanguins, échocardiographiques, et le pronostique des bovins ayant des signes cliniques d'insuffisance cardiaque. Le signalement, l'anamnèse, les signes cliniques, clinicopathologiques, radiographiques et échocardiographiques ainsi que le traitement ont été analyses après une compilation des dossiers médicaux de 59 bovins ayant des signes cliniques d'insuffisance cardiaque et un diagnostic de maladie cardiaque. Le suivi des cas était obtenu par un suivi téléphonique avec le propriétaire. La majorité des patients étaient tachycardes (n = 50), en tachypnée (n = 55). La péricardite d'origine traumatique (n = 21), par extension d'une pleurésie (n = 3), ou idiopathique (n = 1) a été diagnostiquée dans 25 cas. Les autres diagnostics posés étaient une maladie cardiaque congénitale (n = 13), la cardiomyopathie dilatée (n = 9), l'endocardite bactérienne (n = 7) et un processus tumoral (n = 5). Vingt cas (20 %) ont reçu leur congé de l'hôpital. Le taux de survie à long terme était bon pour 2 des 3 cas traités par péricardiostomie. La mort prématurée dans le mois suivant le congé de l'animal (n = 3) ou entre 1 et 3 mois suivant son congé (n = 3) était fréquemment rapporté pour les cas dont où un suivi était disponible (n = 8). Les signes cliniques d'insuffisance cardiaque des bovins sont principalement dominés par des symptômes d'une insuffisance cardiaque droite. Le pronostic est généralement faible chez ces animaux, même si une péricardiostomie peut s'accompagner d'un pronostic favorable chez les bovins souffrant de péricardite septique.

(Traduit par les auteurs)

Introduction

Heart failure (HF) is the terminal event of heart disease (1). Progressive, adaptative changes in the neurohormonal axis develop and have multiple negative effects on the myocardium and cardiac output (2). Clinical signs of HF in cattle are a consequence of increased hydrostatic pressure and peripheral edema (2). The prognosis of heart disease (HD) is poor when clinical signs of HF are observed (1,2). The prognosis of HD in cattle is classically reported to be guarded to poor even if HF is absent (3,4). Only a few clinical studies of cattle with specific HD are available (3–10). Bacterial endocarditis (BE) (5,6), traumatic reticulopericarditis (5,10), lymphoma involving the myocardium (3), dilated cardiomyopathy (9), ventricular septal defect [VSD (4)] and other congenital heart diseases (CHD) (11) are most commonly reported. The clinical signs accompanying HF are multiple. They include peripheral

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edema, pulmonary edema, distended or pulsatile jugular vein, and weakness or exercise intolerance in large animals (2). Cough associated with pulmonary edema, syncope, and cardiac weight loss are signs of HF most commonly described in small animals with a failing heart (1), but are rarely reported in cattle (2). The objective of this retrospective study was to describe the clinical signs, ancillary tests, treatment, and prognosis of cattle diagnosed with heart failure.

Materials and methods

Medical records of cattle presented to the Centre Hospitalier Universitaire Vétérinaire (CHUV) of the Université de Montréal with a final diagnosis of HF or HD from 1990 to 2005 were reviewed. Each animal underwent a thorough clinical examination. The animals retained for this study had to present at least one of the clinical signs of heart failure recognized in other species (1,2) and a definitive diagnosis of heart disease. These clinical signs included weakness, difficulty in walking normally (but without lameness, neurologic, metabolic disease or toxemia) or in standing without help, syncope, dyspnea, peripheral edema, jugular venous distension, increased capillary refill time, and cough not associated with pneumonia (1,2). The animals were excluded from this study if a concomitant noncardiac disease could explain the clinical signs. The final diagnosis of HD was confirmed with echocardiography, clinical signs, and/or necropsy.

For each case, age, sex, breed, duration of clinical signs, and the initial diagnosis or reason for referral were recorded. The physical examination results were compiled. In addition, complete blood (cell) count (CBC), hematocrit, red blood cell and leukocyte count, and fibrinogen determination were compiled when available. A new methylene blue stain was done on smears from samples with anemia to determine the presence of reticulocytes in cases of regenerative anemia. The complete biochemical profile results, which included the determination of urea, creatinine, aspartate aminotransferase (AST), creatine kinase (CK), γ-glutamyl transferase (GGT), total protein, albumin, globulin, and calcium, sodium, potassium and chloride concentrations were included when available.

The echocardiographic, radiographic, and electrocardiographic findings were recorded when available. Pericardiocentesis or thoracocentesis results were noted as well as their cytological examination and the aerobic and anaerobic bacterial culture results. The therapeutic interventions were also recorded as well as necropsy findings in cattle that died or were euthanized at the CHUV. The final diagnosis was classified in one of the following subcategories: exudative pericarditis, BE, cardiac tumor, dilated cardiomyopathy, and CHD. Based on the medical records retrieved for cattle with heart disease and HF, the number of cattle with HF was compared for each cardiac disease subcategory with the number of cases of the same subcategory presented to the CHUV without signs of HF.

Outcomes for all cases discharged from the CHUV were obtained by telephone conversation with the owners 1 to 8 y after discharge. Owners were asked for the productivity of the animal which was defined as adequate or inadequate milk production for adult cows (as judged by the owner) or adequate growth for young cattle when compared with herdmates. Reproduction information and reasons for culling were also investigated.

Statistical analysis

Sex distribution and breed frequency were compared by chi-squared tests between cattle with HF and all cattle referred to the CHUV during the same period. The number of cases of each cardiac disease for cattle having clinical signs of heart failure was compared with cattle without clinical signs of heart failure using the same test. The Kruskall-Wallis test was used to compare the homogeneity of median values of each biochemical parameter and hematologic findings in each group (pericarditis, bacterial endocarditis, congenital heart disease, dilated cardiomyopathy, and tumor). Post-hoc tests were used to find which group was significantly different from the other groups. The level of statistical significance was set at $P < 0.05$.

Results

One hundred and six cattle with a diagnosis of HD were studied. Of the 106 cases, 59 had clinical signs compatible with HF, and 47 had a clinical diagnosis of heart disease without clinical signs of HF. Based on clinical, echocardiographic, pericardial fluid cytology, and necropsy findings, the following subcategories were determined for cases with clinical signs of HF: pericarditis ($n = 25$), CHD ($n = 13$), BE ($n = 7$), dilated cardiomyopathy ($n = 9$) and neoplasia ($n = 5$). During the same period, 47 cases were admitted to the CHUV with a diagnosis of heart disease without clinical signs of HF [pericarditis ($n = 8$), BE ($n = 19$), CHD ($n = 18$), DCM ($n = 1$), and neoplasia ($n = 1$)] (12). The cases of pericarditis and DCM were more frequently presented with clinical signs of HF ($P = 0.006$ and $P = 0.04$, respectively). The cases of BE were more frequently presented without clinical signs of HF ($P = 0.001$). Most animals were female ($n = 57$), Holstein cows ($n = 48$). Other breeds included Ayrshire ($n = 8$), White Belgian Blue ($n = 1$), Limousine ($n = 1$), and Gelbvieh ($n = 1$). During the same period, 11 680 cattle were referred to the CHUV, 10 024 of which were Holstein and 10 573 were female. There were no differences in breed proportion ($P = 0.33$) and gender ($P = 0.11$) when cases with clinical signs of heart failure and the cases referred to the CHUV were compared. The median age at presentation was 4 y (range: 1.5 d to 11.9 y). The reasons for referral were: anorexia ($n = 23$), respiratory problems ($n = 14$), weakness or syncope ($n = 7$), suspicion of heart disease ($n = 6$), presumptive diagnosis of traumatic reticuloperitonitis (TRP, $n = 6$), fever of unknown origin ($n = 6$), gastrointestinal problems ($n = 5$), agalactia ($n = 4$), and lameness ($n = 3$). The median duration of the condition prior to referral was 7 d (from 1 to 250 d, $n = 48$). The stage of production was available for 26 cases: 20 cows were milking (7 calved < 6 wk ago, and 13 had been milking for > 6 wk) and 6 cows were dry in late pregnancy.

Clinical signs of heart failure included brisket ($n = 29$; 49%), submandibular ($n = 19$; 32%), and ventral abdominal ($n = 9$; 15%) edema. Jugular distension ($n = 41$; 69%) or pulse ($n = 26$; 44%) was a common finding. Mammary vein distension was present in 4 cases (7%). Clinical signs indicative of ascites ($n = 5$; 8%) were abdominal distension, splashing sounds when

Table 1. Median values of biochemical parameters and leukocytes in cattle with clinical signs of heart failure secondary to various heart diseases

Blood parameters	All cases	Peri	BE	CHD	DCM	Tum	References used at the CHUV
Fibrinogen	**6**	**6**[a]	**5**	**3**[a]	**3**[a]	**4**	**3–5 g/L**
Total protein	**63.8**	**67.6**	**72.0**[b]	**60.5**	**58.3**[b]	**63.3**	**59.5–80 g/L**
Albumin	27.5	27.3	24.1	29.3	28.4	30.8	27.7–40.4 g/L
Globulin	**35.6**	**40.1**	**47.0**[c]	**32.0**	**24.7**[c]	**29.6**	**26.2–45.2 g/L**
Urea	4.86	4.3	4.69	5.06	7.95	4.70	1.61–6.51 mmol/L
Creatinine	85	85	71	91.5	111	81	54–132 μmol/L
GGT	**53**	**53**	**32**[d]	**36**	**138**[d]	**34**	**9.5–39 IU/L**
AST	**112**	**92**[e]	**129**	**109**	**349**[e]	**126**	**30–104 IU/L**
CK	525	455	360	488	2539	399	0–310 IU/L
Na	138.2	137.3	136.4	141.3	138.6	140.4	134–147 mmol/L
Cl	99.2	97.0	95.4	99.6	96.3	100.3	96–109 mmol/L
K	4.2	4.08	4.18	4.30	4.22	4.33	3.86–5.28 mmol/L
Ca	2.18	2.15	2.13	2.18	2.22	2.15	2.22–2.70 mmol/L
WBC	11.4	13.4	10.1	7.9	10.3	7.5	4.0–12.0 × 1000 cells/μL
Neutrophils	7.61	8.79	6.3	4.8	7.8	4.1	0.6–4.0 × 1000 cells/μL
Lymphocytes	2.38	2.5	2.36	2.9	2.0	2.0	2.5–7.5 × 1000 cells/μL

Bold categories are parameters which showed significant heterogenicity when compared by the Kruskall-Wallis test.
Peri — pericarditis, BE — bacterial endocarditis, CHD — congenital heart disease, DCM — dilated cardiomyopathy, Tum — heart tumor, GGT — gamma glutamyl-transferase, AST — aspartate aminotransferase, CK — creatine kinase, Na — sodium, Cl — chloride, K — potassium, Ca — calcium, WBC — white blood cells.
[a] Fibrinogen was significantly lower in DCM and CHD than in Peri ($P = 0.02$).
[b] Total protein was significantly lower in DCM than in BE ($P = 0.02$).
[c] Globulin was significantly lower in DCM than in BE ($P = 0.003$).
[d] GGT values were significantly lower in BE than in DCM ($P = 0.04$).
[e] AST values were significantly lower in Peri than in DCM ($P = 0.049$).

balloting the abdomen, or transrectal palpation of ascites. A cough not associated with pneumonia as determined by physical examination and ancillary tests ($n = 3$; 5%) and syncope ($n = 2$; 3%) were rarely recorded. The mucous membranes were examined in 58 cases and were normal ($n = 33$; 57%), pale ($n = 20$; 34%), cyanotic ($n = 3$; 5%), or congestive or hyperemic ($n = 2$; 3%). Capillary refill time was less than 2 s in most cases ($n = 49$; 84%).

Cardiac evaluation revealed tachycardia in 50 of 56 cases with a median heart rate of 108 beats/min (range: 54 to 168 beats/min). Abnormal heart sounds were detected in 42 cases and included muffled heart sounds ($n = 23$; 41%), a systolic, diastolic, or systolodiastolic murmur ($n = 17$; 30%), or splashing sounds ($n = 3$; 7%). Dysrhythmia was noted in 7 cases. Respiratory rates were recorded in 55 cases. Tachypnea was present in most cases (med = 43 breaths/min and ranged from 16 to 112 breaths/min). The rectal temperature was recorded in 57 cases with a median of 38.8°C (range: 35.4°C to 40.8°C). Other clinical signs included hypomotility of the rumen in 15 cases (26%), abnormal respiratory sounds consisting of crackles or wheezes in 10 cases (18%), dehydration in 5 cases (9%) and phlebitis in 4 cases (7%).

Results of serum biochemistry profiles and CBCs were available in 54 cases and are summarized by categories of heart disease in Table 1. The median values of fibrinogen, total protein, globulin, AST, and GGT were not similar for each heart disease category. All other blood parameters were not significantly different ($P > 0.05$). The frequent findings in cattle with clinical signs of HF were elevation of gamma-glutamyl transferase (GGT) activity in 33 cases (60%; med = 53 IU/L); aspartate aminotransferase (AST) in 33 cases (60%; med = 112 IU/L); and creatine phosphokinase (CK) in 29 cases (53%; med = 525 IU/L). Complete blood (cell) count revealed anemia (med = 28.4%) in 24 cases (42%). Based on a low hematocrit

and the absence of reticulocytes on blood smears, a nonregenerative anemia was diagnosed in 19 cases (79%).

A pericardiocentesis or thoracocentesis was performed in 21 cases either at the left fifth intercostal space or guided by ultrasound. The findings were compatible with a suppurative inflammation ($n = 11$; 69%), a modified transudate ($n = 2$; 13%), a simple transudate ($n = 2$; 13%), and neoplasia ($n = 1$; 7%). Free or phagocytized bacteria were seen in 4 samples (25%).

Samples that were submitted for bacteriological culture included: transtracheal aspirate ($n = 3$), pericardiocentesis ($n = 21$), blood culture ($n = 5$), or heart tissue taken during necropsy ($n = 8$). Results of bacteriological culture are summarized in Table 2. *Arcanobacterium pyogenes* was the most common bacterium isolated but a mixed flora was commonly obtained.

Electrocardiographic examination was performed in 6 cases and showed sinus tachycardia ($n = 4$), ventricular extrasystoles, atrial fibrillation ($n = 1$), and atrial standstill ($n = 1$). Thoracic radiographs were taken in 36 cases. Observations that were made included cardiac silhouette enlargement in only 3 cases (8%) which had traumatic reticulopericarditis. The most common abnormalities were changes compatible with pneumonia in 19 cases (53%), pleural effusion in 15 cases (42%), and traumatic reticuloperitonitis with a metallic foreign body in proximity to the heart in 9 cases (25%).

Echocardiographic findings were recorded in 51 of 59 cases. Echocardiographic diagnoses were pericardial effusion compatible with fibrinous pericarditis or thickening of the pericardium in 18 cases (35%), pericardial effusion of unknown origin in 11 cases (22%), CHD in 10 cases (20%), and BE in 7 cases (14%). Pleural effusion was diagnosed in 12 cases (24%). Echocardiographic findings of pericarditis were confirmed at necropsy in 18 of 23 cases (78%). Five cases were not classified as pericarditis because of pleural effusion that interfered with the visualization of the pericardium ($n = 2$), and the presence

Table 2. Bacteriological culture in bovine cases of CHF

Sample	Number of cases	Number of samples with culture (%) positive	Bacteria isolated (number)
Pericardial fluid	21	15 (71)	*A. pyogenes* alone (4), or with *F. necrophorus* (2), *B. melaninogenicus* (2), *P. aeruginosa* (1), *E. coli* (1), *Streptococcus* — alpha-hemolytic (1), *M. haemolytica* (2), *H. somni* (1), *Bacillus* spp. (1), *E. coli*, and *P. aeruginosa* (1)
Heart tissue (necropsy)	8	8 (100)	*A. pyogenes* (5), *Staphlococcus aureus* (1), *Acinetobacter* and *Enterococcus* (1), *E. coli*, *Streptococcus bovis*, *Enterococcus* (1)
Blood culture	5	1 (20)	*A. pyogenes* (1)
Transtracheal aspirate	3	1 (33)	*Moraxella bovis, P. multocida,* and *Mycoplasma bovis* (1)

of pericardial effusion without any inflammatory signs (hyper-echoic material in the pericardial fluid, thickening of the pericardium, *n* = 3). Congenital heart defects were correctly diagnosed in 8 of 11 cases, dilated cardiomyopathy in 7 of 7 cases, and BE in 4 of 6 cases. However, echocardiography could not specifically identify cardiac neoplasms. At echocardiography, neoplasms were associated with pericardial effusion in 3 cases of lymphoma. A mass in close proximity to the aorta and pulmonary arteries possibly exerting a compression on these vessels was observed in 2 other cases.

Of the 59 cattle with clinical signs of HF, 34 (58%) received therapy for their condition. The remaining animals either died or were euthanized without treatment. Treatment included antimicrobial drugs in 26 cases [procaine penicillin (*n* = 13), ceftiofur (*n* = 3), oxytetracycline (*n* = 3), a combination of sodium ampicillin and trimethoprim sulfadoxin (*n* = 3), or procaine penicillin and trimethoprim sulfadoxin (*n* = 2), spectinomycin (*n* = 1) or florfenicol (*n* = 1)], diuretic [furosemide (0.5 to 2 mg/kg, IV or SQ) in 9 cases], intravenous fluids (*n* = 5), pericardial lavage (*n* = 4), pericardiostomy and fifth rib resection (*n* = 3), vitamin E/selenium injection (*n* = 3) or rumenotomy (*n* = 2). Only 15 animals were discharged from the CHUV. Of these, 3 were slaughtered and 12 went back to the farm with a diagnosis of pericarditis (*n* = 6), congenital heart disease (*n* = 5), and cardiac localization of enzootic lymphoma (*n* = 1). Only 8 cases were available for long-term follow-up; 2 of which had a good long-term prognosis. Both had traumatic pericarditis treated by fifth rib resection and pericardiostomy. Three cows were culled 1 to 3 mo following discharge from the hospital, and 3 others died or were euthanized within 1 mo after discharge because of recurrence of the clinical signs or a worsening general condition.

Necropsies were performed on 44 animals after euthanasia (*n* = 35) or natural death (*n* = 9). Seventeen cases had postmortem findings compatible with pericarditis. Causes for the pericarditis were: traumatic (*n* = 13), extension of pleuropneumonia (*n* = 2), pneumonia (*n* = 1), or unknown (*n* = 1). Bacterial endocarditis was observed in 7 cases and affected the tricuspid valve (*n* = 4), pulmonary valve (*n* = 3), mitral valve (*n* = 1), and right ventricular endocardium (*n* = 2). Three cases had bacterial vegetations at more than 1 site. Pleural effusion was observed in 3 cases. In 1 animal, the tricuspid valve, pulmonary valve, and mural endocardium of the right ventricle were affected in association with an atrial septal defect (ASD). Congenital heart disease was observed in 8 animals. Ventricular septal defect (VSD) was the most commonly encountered anomaly (*n* = 6). In 1 animal, Eisenmenger's complex and pulmonary hypertension were also observed. The size of the VSD varied from 2.2 to 5.1 cm. A 6-year-old Holstein cow had clinical signs of HF, a 5.1-cm VSD, and a productive life in the herd until HF occured. Other congenital defects included ASD (*n* = 2) and a patent ductus arteriosus (*n* = 1). Dilated cardiomyopathy was observed in 8 animals.

Histopathological observations included coagulation necrosis of the myocytes in 7 animals and lymphocytic infiltration in 1 animal (9). In 6 of 8 animals, generalized cardiac dilation was present. Four heifers also had disseminated necrosis of the striated muscles (tongue, diaphragm, and limb muscles). Neoplastic lesions involving the heart were observed in 4 animals. In 2 animals, cardiac localization of bovine enzootic lymphoma was identified. In 2 other animals, a voluminous heart base tumor was observed which appeared to compress the large vessels. Histopathological examination of these masses was compatible with a tumor of the mediastinal fusiform cells. Other necropsy findings that were observed in the animals with HF were: various degree of peripheral edema (*n* = 44; 100%), liver congestion with variable hepatocyte necrosis (*n* = 13; 30%), ascites (*n* = 6; 14%), hydrothorax (*n* = 5; 11%), pulmonary edema (*n* = 4; 9%), and renal necrosis (*n* = 2; 5%).

Discussion

A retrospective study of clinical HF in cattle has not been previously reported. One may debate the criteria for definition of cases with HF in this study since there was no objective assessment of ventricular function in most cases. The syndrome of HF is defined as a ventricular dysfunction leading to inability to maintain an adequate blood output (13). Inadequate cardiac function leads to a myriad of symptoms (1,13); however, many of these symptoms can be caused by noncardiac diseases. This was the main reason why, in order to avoid cases without the syndrome of HF, we selected medical records with clinical signs of HF and a diagnosis of HF secondary to a primary cardiac disease as previously reported in an equine retrospective study (14). Cases with a noncardiac disease that could explain the clinical signs of poor perfusion and edema (such as endotoxemia or sepsis) were excluded. These selection criteria were used in the present study and are representative of the clinical presentation of the syndrome of HF in cattle. A prospective study still needs

to be conducted to document ventricular function in bovine heart failure.

The clinical picture of the syndrome in bovines appears to be different from that in companion animals or horses, in which the predominant clinical signs were associated with left-sided heart failure (1,14). Right-sided heart failure, manifested by peripheral edema and distension of the venous system, was the most common finding in cattle. Left-sided heart failure overloads the pulmonary circulation and causes respiratory symptoms such as dyspnea, and cough (1). Although complaints involving the respiratory system were the primary reasons for referral in 14 of 59 cases, pulmonary edema or congestion was rarely found. Most of the respiratory problems were associated with pneumonia or pleural effusion. In fact, pulmonary edema was identified in only 4 of 44 animals that had been necropsied. The absence of pulmonary edema may indicate that the left heart is rarely involved in HF in bovine species, in contrast to companion animals (1). Also, in a retrospective study of equine species, left-sided heart failure associated with cough, dyspnea, and pulmonary edema was only present when mitral regurgitation occurred (14).

Of the 5 categories of cardiac disease that were characterized in this study (pericarditis, BE, CHD, cardiomyopathy, and neoplasm), pericarditis ($n = 33$), CHD ($n = 31$), and BE ($n = 28$) were the most common, while dilated cardiomyopathy ($n = 10$) and cardiac neoplasia ($n = 6$) were found less frequently. When comparing the proportion of animals with HF to those without HF, one can distinguish 2 types of heart disease. The first type was more frequently associated with signs of HF. Cattle with pericarditis and dilated cardiomyopathy showed signs of HF more frequently. By contrast, BE was less frequently accompanied with HF.

Differences in the frequency of the clinical manifestations of HF could be due to differences in the pathophysiology of the diseases. Pericarditis and pericardial effusion progressively lead to decreased diastolic filling of the right ventricle and, eventually, to tamponade (15). The right ventricle is initially more affected because it is much more vulnerable to increased pericardial pressure, causing the right ventricular telediastolic volume to fall (15). This leads to retrograde increased venous pressure and peripheral edema. In the case of dilated cardiomyopathy, there is a dramatic decrease in systolic function (2). Echocardiography revealed left and right ventricular dilation and decreased shortening fraction (16). The decreased filling and systolic function lead to decreased right cardiac output (1). Finally, in cases where a heart base mass is compressing the basal portion of the heart, such as a mediastinal fusiform cell tumor, increased retrograde venous pressure leads to peripheral edema without major heart dysfunction.

By contrast, BE was less frequently associated with signs of HF. Valvular bacterial endocarditis may disturb the closure of the affected valve (17). This is accompanied by regurgitation that may cause cardiac remodeling such as dilation and hypertrophy (17). Congestive heart failure is the final stage of chronic and severe BE, creating severe myocardial damage or remodelling (6,17,18).

Congenital heart diseases in cattle can consist of multiple malformations (11). The most commonly encountered syndromes are VSD, ASD, and tetralogy of Fallot (11). The first 2 rarely cause HF, which is secondary to a flow volume disorder caused by the disturbed blood flow through these defects (9). In the cases of tetralogy of Fallot, the most common clinical symptoms are cyanosis, polycythemia, and poor growth (19,20). Hypoxemia can contribute to damage to the myocardium that could ultimately cause HF.

Serum biochemistry results were compatible with muscle (AST, CK), or liver (AST, GGT) damage; however, no specific tests for cardiac cell damage were done. Cardiac isoenzyme of lactic dehydrogenase (LDH1), myocardial bound CK, or cardiac troponin I (cTnI) have a higher specificity for myocardial damage in other species. The cTnI is a highly sensitive test recently used to document myocardial dysfunction and response to therapy in 1 bovine case of idiopathic pericarditis and cardiac tamponade (21). This ancillary test can be done using human radioimmunoassay kits because of the highly similar sequence of amino acids in human and bovine cTnI (22). The serum cTnI levels may be increased in cattle secondary to traumatic pericarditis (23,24), bacterial endocarditis (24,25), congenital heart defects (24), as well as other thoracic diseases (24). In other species, the determination of cTnI levels may become a prognostic indicator when HF is diagnosed (26,27).

Other results of blood tests were compatible with an inflammatory process that was attributable to cardiac disease (BE, pericarditis) or to other inflammatory complications (pneumonia in the case of CHD). A chronic inflammatory process was observed more often in cases of BE, which also had the highest values for globulins. This disease is frequently associated with other extracardiac chronic inflammatory processes (musculoskelettal infection, chronic mastitis, or metritis) (5) which can precipitate thromboembolization to the endocardium (28).

Therapeutic attempts and successes were rare in this study. Little is known of the efficacy of the treatment for HF and most available veterinary drugs have no homologation for cattle and no specific withdrawal period. Therapy should be symptomatic or rarely specific for certain forms of heart disease. Peripheral edema and jugular distension are classically treated by repeated administration of furosemide (29). The objective is to reduce cardiac preload which will eventually decrease congestion and edema. Also, electrolyte balance must be assessed to avoid complications associated with hypokalemia, hyponatremia, and hypocalcemia which could develop with treatment (30). Finally, malignant arrhythmias should be investigated and treated if necessary (29,30).

Antibiotics (preferably based on sensitivity of the bacteria isolated after blood culture) should be administered to avoid the progression and dissemination of the infection when BE is diagnosed (28,31). The efficacy of this treatment remains to be studied in a large group of animals. In 1 case report, a survival of 14 mo was observed (31). Two retrospective studies reported survival rates of 27% ($n = 33$) (32) and 18% ($n = 22$) (6) between 6 to 20 mo after presentation. In the latter study, long-term survivors were younger (more than 6 mo of survival, mean age of 14 mo) than those that died, were euthanized, or were short-term survivors (mean age of 38 mo). However, it was not noted whether or not survivors had been presented with or

without signs of HF. Cases with BE associated with CHF have a poor prognosis (32). None of the 7 cows in our study that had BE and HF were discharged. Only 1 cow was salvaged. Four animals were euthanized and 2 cows died during their hospitalization despite initiation of treatment.

Pericardial disorders in cattle are most commonly associated with complications of TRP (33). Idiopathic pericarditis has recently been described (21,34,35). The correct diagnosis of this rare condition by means of pericardiocentesis is important since the therapeutic success rate seems to be good to excellent with pericardial drainage and lavage (21,34,35). Unfortunately, in our study, the cow with pericardial findings suggestive of idiopathic pericarditis was euthanized without any therapeutic attempt; necropsy did not reveal any evidence of a traumatic origin of the pericarditis. The treatment of pericardial diseases classically consists of pericardial drainage and lavage (21,33) or pericardiostomy, if the effusion was considered too viscous (presence of clots) (36,37). The prognosis was guarded because of the risk of constrictive pericarditis (32). In the present study, 2 of 3 cases of pericarditis treated by pericardiostomy and lavage had a favorable outcome. Both heifers were carrying valuable embryos and had normal pregnancies and calving. Adequate milk production was observed for only 1 of them. The longest time reported after pericardiostomy was 6 y and 5 lactations (36). The third heifer that was treated by pericardiostomy died 3 mo after being discharged without having a productive life and was therefore considered as a negative outcome. None of the patients that were treated by simple pericardial lavage and drainage had a favorable outcome in the present study. This procedure should be used only for acute cases or nonseptic pericarditis (21,34,38).

Congenital heart disease is associated with a poor prognosis when signs of HF are present (9,39–42). In the present study, all calves diagnosed with HF due to CHD had a poor outcome. Congestive heart failure appears to be frequently accompanied by severe myocardial dysfunction (9). This was observed in a red Holstein adult cow with a large VSD showing signs of HF. This cow had previously calved 4 times and had had a normal productive life in the herd prior to illness. Necropsy findings, however, showed myocardial necrosis and fibrosis that could also be interpreted as early lesions of secondary dilated cardiomyopathy. The clinical signs of HF in this case were most probably associated with decreased capacity of the myocardium to compensate for the VSD and its associated hemodynamic disturbances by developing dilated cardiomyopathy.

Dilated cardiomyopathy may be primary with a genetic origin in red Holstein (8), and Ayrshire cattle (43,44). Secondary cardiomyopathies are principally caused by intoxication (ionophores such as monensin) (45), vitamin E/selenium or copper deficiency, viral or bacterial infection, or any other debilitating cardiac disease (2). Most cases reported in this study had suspected primary dilated cardiomyopathy affecting mostly red cattle (6 Ayrshire, 3 red Holstein) (43,44). Coagulation necrosis found in the myocardium and exposure to monensin in the feed in 5 animals, however, did not rule out the possibility of secondary cardiomyopathy due to ionophore intoxication (45). No specific treatments for these 2 myocardial disorders exist.

Correction of electrolyte imbalances is of primary importance (2,29,46). The use of positive inotropes could be considered in valuable animals. The inotropic drugs (digoxin, for example) should be used with care due to their side effects (2). A constant monitoring of the hydration status, acid-base, and electrolytes imbalance is therefore required. Inotropic drugs are also contraindicated in cases of monensin toxicity (2); however, to the authors' knowledge, there are no reports of long-term survival in bovine cases of HF secondary to cardiomyopathy (2,8,44–47).

Heart tumors were fatal in all cases because no specific therapy was available (29). In 1 case of cardiac lymphoma, the animal was symptomatically treated with furosemide but died 1 mo later. As shown in a previous study, degradation of the general health was rapid once systemic signs occured (decreased milk production, anorexia, HF) (3). Loss of production associated with clinical signs, malignancy of neoplasm, and the absence of safe antitumor drug therapy for food animals do not allow the clinician to consider long-term control of the disease.

Most of the clinical signs of congestive heart failure in cows resulted from right-sided heart failure with peripheral edema, and jugular vein distension or pulse. Results in this study also suggest that prognosis for cows with clinical signs of congestive heart failure and heart disease is poor. This is in agreement with other literature. Bacterial endocarditis, congenital heart disease, cardiac neoplasms, and cardiomyopathy had a poor prognosis when signs of HF were seen. Traumatic pericarditis, despite manifestations of HF, when aggressively treated with pericardiostomy can produce acceptable outcomes. The cost of this treatment and need for long-term antibiotic therapy, however, allow this treatment only in highly valuable animals. CVJ

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